

Slow waves and sleep regulation.

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It was recognized early on that sleep intensity is reflected in the sleep electroencephalogram (EEG) by the prevalence of low frequency, high amplitude waves called slow waves. Under normal physiological conditions, slow waves in the non-rapid eye-movement (non-REM) sleep EEG can be regarded as an indicator of "sleep depth" or "sleep intensity".

The term "slow waves" is often used in a rather loose way in the literature and typically comprises different rhythmic components like slow oscillations (< 1 Hz) and delta activity (1 – 4 Hz). At the cellular level slow oscillations consist of sequences of depolarized (up states; neuronal activity) and hyperpolarized (down states; neuronal silence) components and at the level of multi-unit activity recorded in the cortex, slow oscillations are reflected in sequences of "on" (spiking) and "off" (no spike activity) periods.

To examine regional aspects of slow waves, we used source modeling and topographic maps to locate and display activation of brain structures in the 0.5 - 2 Hz frequency range during slow wave sleep under normal conditions and after sleep deprivation in the first non-REM sleep episode (where sleep pressure is highest). Power maps revealed a frontal predominance of all frequencies between 0.5 and 2 Hz. Furthermore, an occipital focus of activity was present below 1 Hz. Maps at 1 Hz and below were not affected by sleep deprivation, whereas an increase in power was present in the maps above 1 Hz. Based on the response to sleep deprivation, low- and mid-delta activity (0.5 - 1 Hz; 1.25 - 2 Hz) were dissociated. Electrical sources within the cortex revealed a predominantly frontal distribution of activity for both low- and mid-delta activity. Sleep deprivation resulted in an increase in source strength for mid-delta activity only, mainly in parietal and frontal regions. A salient aspect of sleep is that it is homeostatically regulated. Sleep pressure builds during the day and dissipates across the night (homeostatic Process S). A sleep deficit results in the prolongation and intensification of sleep during subsequent sleep. An important physiological marker of non-REM sleep pressure is EEG spectral power in the delta band (sleep EEG power between 0.75 and 4.5 Hz), also referred to as slow-wave activity (SWA). SWA shows a rebound following sleep deprivation proportional to the amount of sleep lost. Moreover, SWA declines across the sleep episode. Process S can be modeled as a saturating exponential function during wakefulness and an exponential decline during sleep. Considerable individual variation in the parameters of Process S have been observed.

We examined whether the dynamics of sleep homeostasis (i.e., time constants of buildup and decline of Process S) change across early development in a longitudinal study (at 2, 3 and 5 years of age). Time constants of the buildup increased with increasing age while the time constants of the decline were similar across ages. We observed a faster increase of Process S in early pubertal children compared with mature adolescents, while the decrease of S was similar in both groups (cross sectional study). The increased maturational increase in the time constant of the build-up may reflect an increased tolerance to sleep deprivation

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